

# Hedonic Eating Predicts Fat Mass in Children with Healthy-weight at Low Familial Risk for Obesity

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## Introduction

- Increased adiposity in children is associated with food responsiveness and non-homeostatic eating (i.e., eating in the absence of hunger-EAH)<sup>1,2</sup>
- The extent to which familial risk for obesity moderates the association between EAH and adiposity is not known
- The objective of these analyses was to determine how EAH and familial risk for obesity relate to adiposity among 7-8-year-old children

## Hypotheses

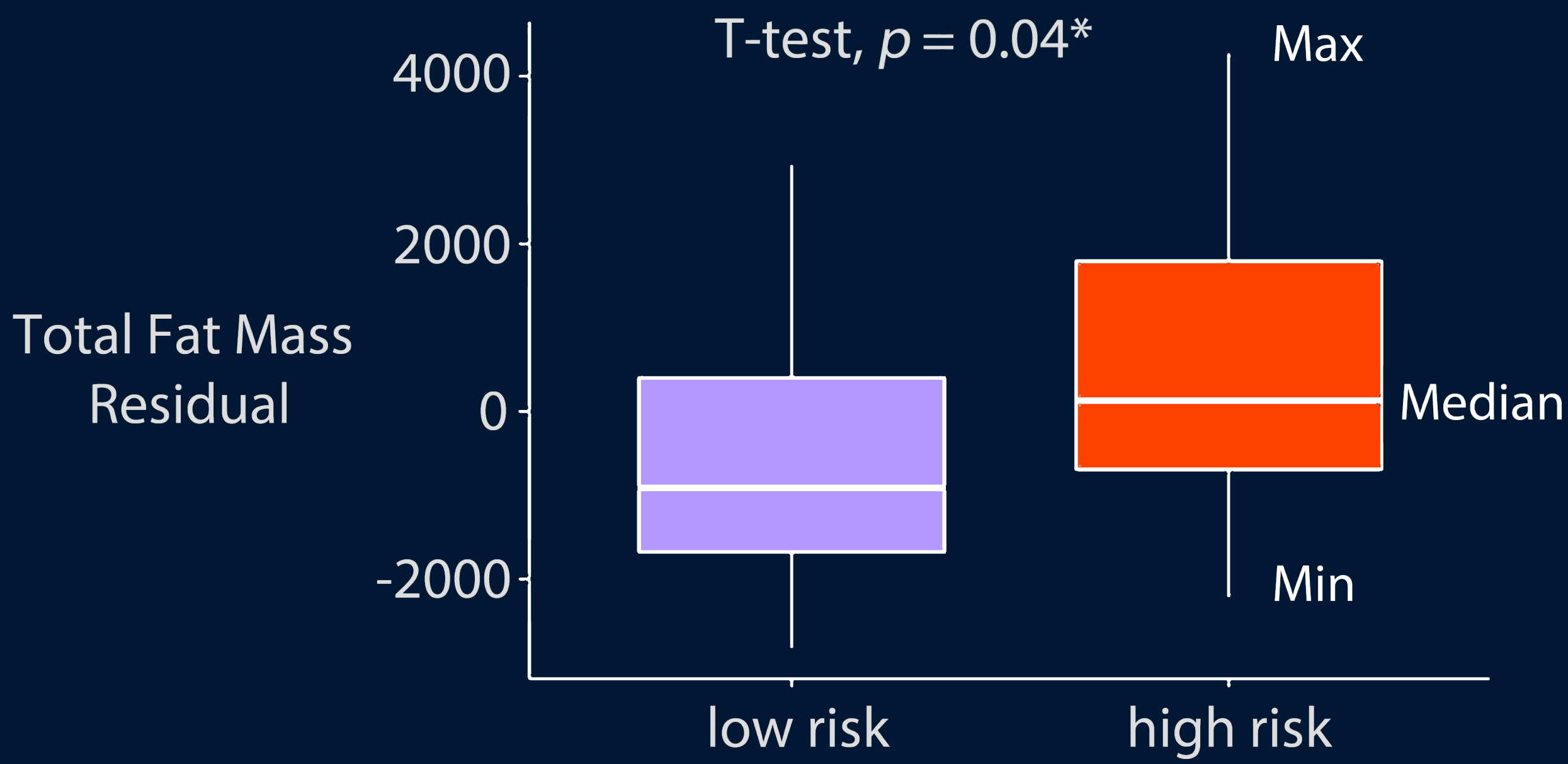
- Hypothesis 1:** EAH would be associated with greater fat mass
- Hypothesis 2:** This relationship would be stronger among children at high familial risk for obesity based on predisposition for increased adiposity

## Participants

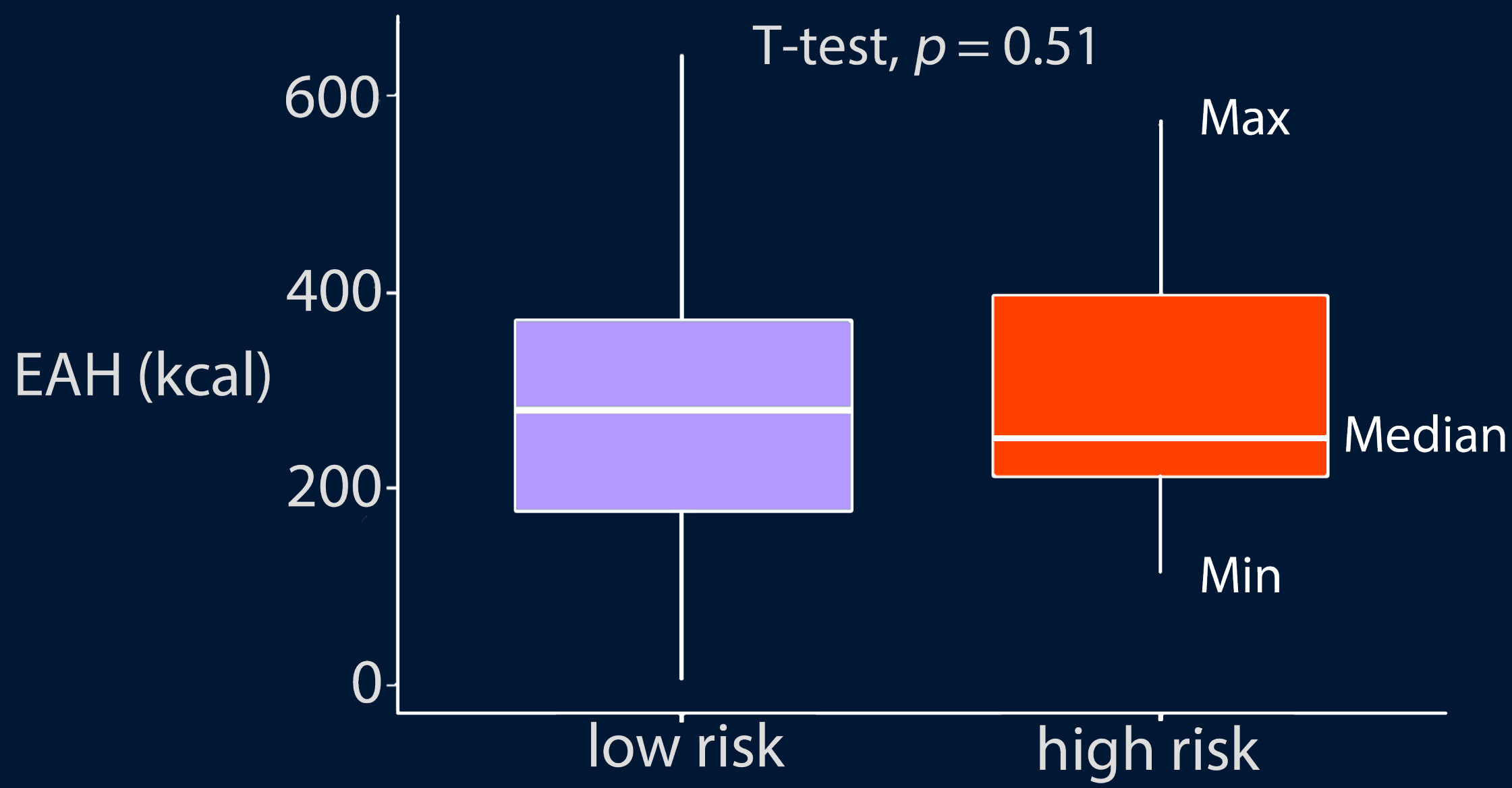
	Low Familial Risk (N = 33)	High Familial Risk (N = 24)	p
Sex, N			0.46
Male, N	14	9	
Female, N	19	15	
Age, years (SD)	7.87 (0.61)	8.04 (0.58)	0.30
BMI, percentile	43.70 (26.45)	57.37 (19.40)	<b>0.03*</b>
Fat Mass, grams (SD)	6983.82 (1630.49)	8180.38 (1905.82)	<b>0.02*</b>
EAH, kcal (SD)	285.65 (141.27)	310.72 (142.86)	0.51
Race, N			0.58
Caucasian	32	24	
Asian	1	0	
Household Income, N			0.14
>\$100,000	14	4	
\$50,000-\$100,000	14	13	
<\$50,000	4	6	
Not Reported	1	1	

**Table 1.** t-tests were used for Age, BMI percentile, and fat mass. Fisher’s exact test was used for sex and race and X<sup>2</sup> was used for household income.

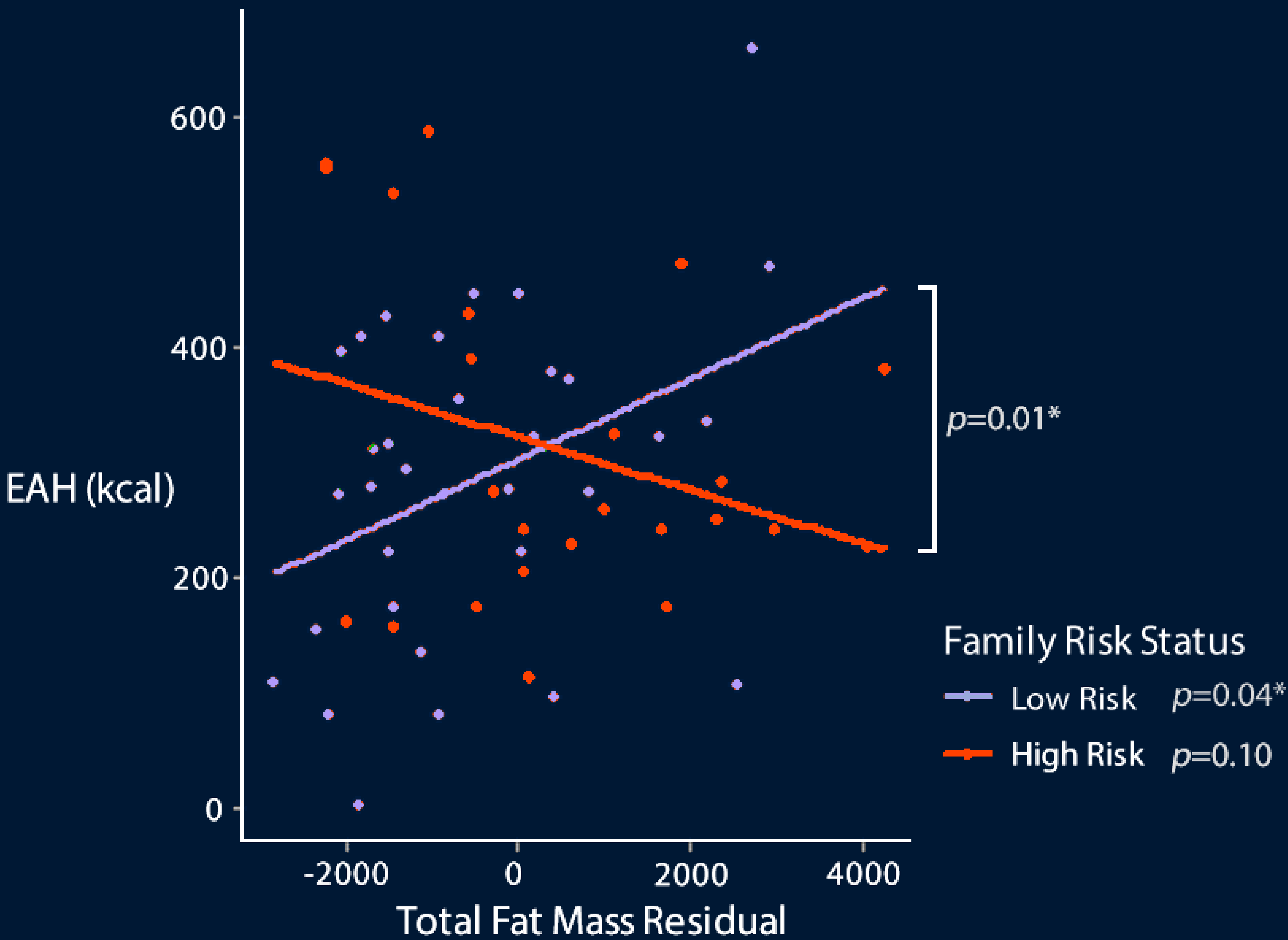
# Familial risk status for obesity moderates the relationship between fat mass and EAH in children with healthy-weight



**Figure 1.** Children with high familial risk had greater fat mass compared to low familial risk children



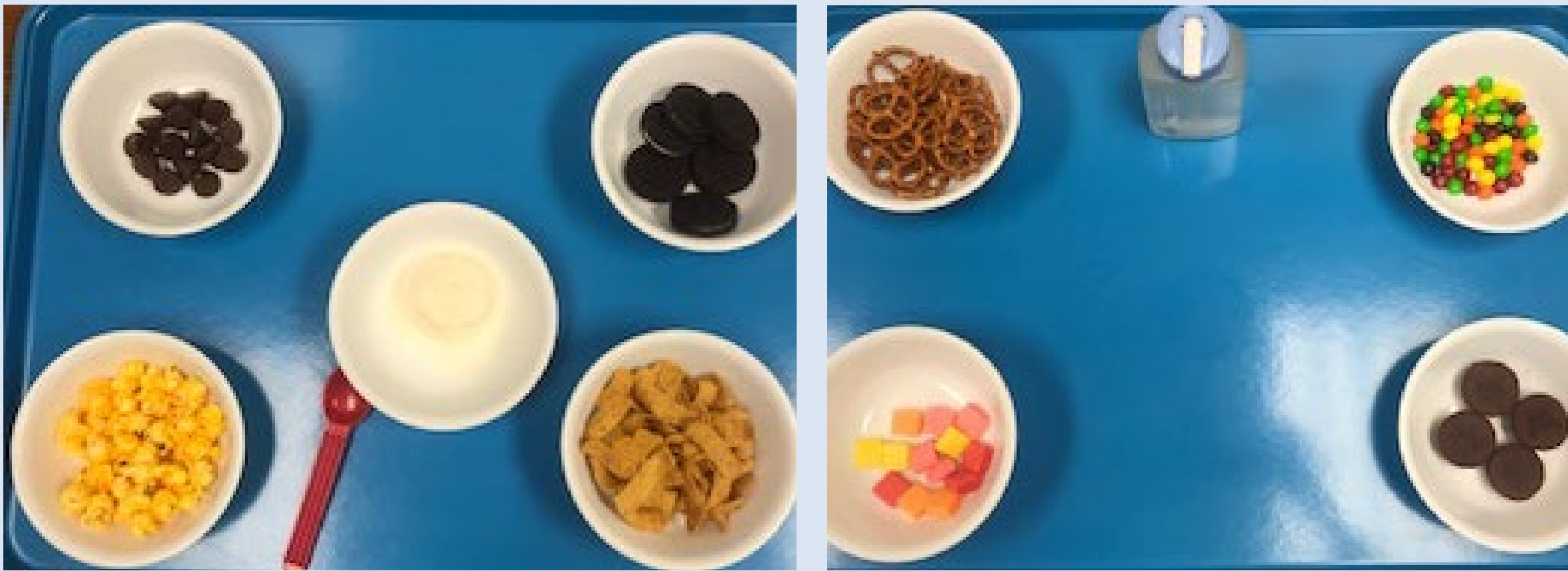
**Figure 2.** Familial Risk status was not independent associated with EAH intake



**Figure 3.** Linear regression model assessing the interaction of familial risk status on the relationship between EAH (kcal) and fat mass residual (controlled for sex and age). High EAH in low risk children was significantly associated with increased fat mass  $p=0.04$ . The opposite trend was seen in high risk children but was not significant  $p=0.10$ .

## Methods

- Preliminary data analysis from an ongoing longitudinal study
- Familial risk for obesity was determined by parental BMI ( $\text{kg}/\text{m}^2$ )<sup>3</sup>
  - Low-risk (both parents: BMI < 25)
  - High-risk (Mother: BMI > 30, Father: BMI > 25)
- Child fat mass (grams) was assessed with dual x-ray absorptiometry
- Following an ad-libitum multi-item meal, children were presented with an array of snack foods and toys/games and given 10 min to eat from these foods
- Intake was assessed by measuring pre- and post-weight



**Figure 4:** Snack foods served during EAH protocol

## Results

- Familial risk for obesity was associated with child fat mass ( $p<0.01$ ) such that high-risk children had greater adiposity
- No main effect of EAH on adiposity, however, familial risk for obesity interacted with EAH to significantly influence EAH ( $p=0.01$ )
- EAH was positively associated with fat mass among children at low-risk, but not high-risk for obesity

## Conclusions

- Contrary to our hypotheses, EAH was positively associated with greater adiposity in healthy-weight children who were at low, not high, familial risk for obesity
- Children at high familial risk for obesity who remain healthy weight may have a protective phenotype for obesity

## References

<sup>1</sup>Carnell, S., & Wardle, J. (2008), Am J Clin Nutr, 88(1), 22-29.  
<sup>2</sup>Fearnbach, S. et al. (2015), Appetite, 92, 233-239.  
<sup>3</sup>Whitaker, K.L. et al. (2010). Am J Clin Nutr 91, 33-39.

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